Magnetic Resonance Imaging of Rhabdomyolysis: Muscle Necrosis Versus Ischemia

Yu-Ching Cheng  Howard Haw-Chang Lan  Chun-Hsi Shih  Clayton Chi-Chang Chen  San-Kan Lee

Department of Radiology, Taichung Veterans General Hospital, Taichung, Taiwan

ABSTRACT

Rhabdomyolysis is a syndrome caused by injury to skeletal muscle involving leakage of large quantities of potentially toxic substances into plasma made by laboratory evaluation. Although rhabdomyolysis is seldom discussed on magnetic resonance imaging (MRI), the specific presentation of rhabdomyolysis on MRI can be used as a tool in differencing various etiologies of myopathies but also to evaluate the distribution and extent of injury of affected muscles. Two distinct imaging types of rhabdomyolysis can be distinguished in the initial of diagnosis, i.e. type 1, homogeneous signal changes and contrast enhancement in the affected muscles, signified an ischemic or reversible ischemic reaction, and type 2, homogeneous or heterogeneous signal changes and rim enhancement, and might presence of “stipple sign” in the affected muscle on contrast-enhanced images, signified an irreversible muscular necrosis. However, to our best knowledge, the imaging-histological correlation that indicated the affected muscles were reversible (ischemia) or irreversible (necrosis) was not reported. We present a case of rhabdomyolysis demonstrating both typical type 1 and type 2 signal changes on the initial MRI, and complete remission of signal changes in type 1 lesion (ischemia), and persistence of type 2 lesions (necrosis) on follow-up MRI after 3 months and 6 months.

Rhabdomyolysis is a condition in which rapid breakdown of skeletal muscle fibers resulting in leakage of potentially toxic cellular contents, including electrolytes, myoglobin, and other sarcoplasmic proteins, into the systemic circulation. According to the severity of muscular injury, the clinical presentation may be variable, typically includes muscle pain, weakness and dark urine. The diagnosis is mainly made by elevation of serum creatine kinase (CK) [1, 2]. Although there is no definite cut-off value that conclusively diagnoses rhabdomyolysis, a serum CK activity greater than five times of the upper limit of normal value (60-400 IU/L) was accepted by most of the authors as a criterion for the diagnosis [2]. Magnetic resonance imaging (MRI) was not regarded crucial in the diagnosis of rhabdomyolysis due to the non-specific findings of muscular injury or myopathy from mild degree muscular edema to more severe stage of myonecrosis [3]. MRI was used for evaluating the distribution and extent of the affected muscles. Two typical types of MRI features of rhabdomyolysis had been recently reported, i.e. type 1, homogeneously isointense to hyperintense on T1-weighted, homogeneously hyperintense on T2-weighted and STIR images, and homogeneously enhanced on contrast-enhanced MR image, and type 2, homogeneously or heterogeneously hyperintense on T1-weighted images, heterogeneously hyperintense on T2-weighted images, rim enhanced on contrast-enhanced MR images with “stipple sign” [3]. To our best knowledge, however, there is no literature dealing with the imaging results of rhabdomyolysis. Here in, we would present a case having these two characteristic imaging features on initial MRI, but showed different results on series follow-up MRI.

Correspondence Author to: Howard Haw-Chang Lan
Department of Radiology, Taichung Veterans General Hospital, Taichung, Taiwan
No.1650, Sec. 4, Taiwan Boulevard, Taichung 407, Taiwan
CASE REPORT

A 28-year-old man was buried with tones of sands and stones for hours in an accident of routine work. He was sent to our emergency department and complained bilateral lower legs weakness and numbness. On physical examination, generalized swelling and tenderness of his left leg were noted. Serum CK was 45045 U/L and rhabdomyolysis was diagnosed.

Emergent fasciotomy to the left lower leg at that time was performed for suspicious of compartment syndrome. MRI was performed 10 days after the operation for further evaluation. The images disclosed two distinct imaging abnormalities. First, homogeneous signal changes on both T1WI and T2WI with enhancement in the muscles of the left lower limb, that included the left soleus muscle, quadriceps femoris muscles, etc. (Fig. 1), and second, homogeneous or heterogeneous signal changes on T1WI and T2WI and rim enhancement, and presence of the typical “stipple sign”, demonstrated as dot-like or linear streaky enhanced

**Figure 1**

![Image 1](image1.png)

*Figure 1. Initial MRI of left thigh: Type 1 rhabdomyolysis. a. Axial T1-weighted MR image with fat saturation shows homogeneous hypointensity in left vastus muscle groups (arrows) b. Axial T2-weighted MR image with fat saturation show homogeneously hyperintensity (arrows) c. Axial T1-weighted post contrast MR image with fat saturation shows homogeneous enhancement (arrows).*

**Figure 2**

![Image 2](image2.png)

*Figure 2. Initial MRI of left lower leg: Type 2 rhabdomyolysis. a. Axial T1-weighted MR image with fat saturation shows heterogeneous hyperintensity in anterior and posterior compartment muscles of left lower leg (white arrows) b. Axial T2-weighted MR image with fat saturation show heterogeneous hyperintensity (white arrows) c. Axial T1-weighted post contrast MR image with fat saturation shows rim enhancement with “stipple sign” (black arrowheads).*
foci within an area of rim enhancement in the affected muscles, the anterior and posterior compartments of the left lower leg (Fig. 2). He received intensive medical care and recovered gradually. On series follow-up MRI performed 3, 6 and 30 months after the accident, disclosed the type 1 lesions disappeared (Fig. 3). On the contrary, in type 2 lesions presented persistent constant or dark signal intensity on T1WI and T2WI without contrast enhancement, and decreased muscle bulk (Fig. 4), indicating of intramuscular fibrosis and hemosiderin deposition resulting from previous intramuscular damages and hemorrhage.

**DISCUSSION**

Rhabdomyolysis is a clinical and biochemical syndrome with the definitive diagnosis made by typical clinical symptoms and laboratory evaluation of serum CK [2, 4, 5]. Rhabdomyolysis was caused from congenital or acquired diseased or physical or non-physical factors, including excessive exercise, crush injury, artery occlusion, body temperature changes, metabolic disturbance, drugs and toxins, infection, inflammation and genetic disorders [5]. A markedly elevated serum CK is the hallmark of rhabdomyolysis. The released of toxic substances after muscular injury may result in microvascular damage, capillary leak and increased intra-compartment pressures, reduced tissue perfusion and ischemia.

The most important and primary goal of treatments is to prevent any possible causes that may lead to acute renal failure, such as volume depletion, myoglobinuria or aciduria-related tubular necrosis. Aggressive volume

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**Figure 3**

3a 3b 3c 3d 3e 3f

*Figure 3.* Type 1 rhabdomyolysis series of MRI follow-up: (a, b): initial, (c, d): 3 months, (e, f): 6 months (a, c, e): Axial T1-weighted MR images (b, d, f): Axial T2-weighted MR images with fat saturation

The initial MR images show homogeneous isointense to slightly hyperintense on T1WI and homogeneous hyperintense on T2WI (arrows) recovered to normal muscle signal intensity in serial MRI follow-up at 3 months and 6 months (arrows). The type 1 lesion is completely recovered.
resuscitation plays a critical role in the initial supportive care [2, 5]. Another major therapeutic goal is control of electrolyte imbalance, esp. hyperkalemia, for its high possibility of fetal cardiac arrhythmia or arrest. It could be the consequence of rhabdomyolysis or the sequel of acute renal failure. If necessary, dialysis is needed if medical control is not enough for overt hyperkalemia or persistent acute renal failure. Of course, the managements of causative etiologies such as infection, trauma, drugs or toxins to stop the ongoing muscle destruction were mandatory.

Compartment syndrome may declare as an early or late complication in rhabdomyolysis, and should be treated with immediate fasciotomy for decompression and prevent further muscular damage from ischemia and infarction. In general, when the intracompartmental pressure exceeds 30 mm Hg, a fasciotomy is advocated. To accurately localization of the affected muscle groups and the possible damage extension, MRI is the choice of image modality to offer information to the clinician for better pre-operative planning [2, 6].

The prognosis of rhabdomyolysis depends mainly

Figure 4. Type 2 rhabdomyolysis after series of MRI follow-up:
(a, b, c): initial, (d, e, f): 3 months, (g, h, i): 6 months
(a, d, g): Axial T1-weighted MR images
(b, e, h): Axial T2-weighted MR images with fat saturation
(c, f, i): Coronal T2-weighted MR images with fat saturation
The initial MR images (a, b, c) show heterogeneous hyperintense on both T1WI and T2WI at left lower legs including anterior and posterior compartment (arrows). Gradually decreasing signal intensity in affected muscles are observed after series of MRI follow-up at 3 months (d, e, f) and 6 months (g, h, i). Type 2 lesion is caused from myonecrosis, intramuscular fibrosis and/or old hemorrhage that is not reversible.
MRI of rhabdomyolysis

Upon the underlying etiology and whether any morbidity existence, i.e. acute kidney injury or electrolyte imbalance. Rapid appropriate supportive treatment of rhabdomyolysis induced renal impairment can improve the outcome especially in traumatic crush injuries [2].

MRI plays the important role in localization and assessment of the extent of the affected muscles. In addition, it might be helpful to assess the possible reversible and irreversible changes of the affected muscles for treatment planning and rehabilitation programs afterward. MRI features of rhabdomyolysis in affected muscles showed hyperintensity on T2-weighted and STIR imaging, reflecting increased water content possibly due to edema, necrosis or infarction of muscles [7]. The muscles with high signal intensities on T1-weighted images may represent the presence of methemoglobin after intramuscular hemorrhage, fat or proteinaceous content. Nevertheless, these MRI findings of rhabdomyolysis were thought to be non-specific in the past [7-9]. Two distinct MRI features of rhabdomyolysis were proposed in the recent article [3], i.e. type 1 and type 2 muscular signal changes. Histologically, the type 2 lesions were due to myonecrosis of the affected muscles. The type 1 lesions, although had no histological correlation, were thought to be mostly due to overexertion, represent edema of the affected muscles in the initial stage of rhabdomyolysis. Based on MRI only, it might be difficult to be differentiated the type 1 lesions from myositis or myopathy of different causes, i.e. muscle tear, inflammation, infections, etc [7]. However, the clinical history and symptoms with laboratory findings are helpful in differential diagnosis.

The recovery of type 1 rhabdomyolysis may depend on local reperfusion of muscles. Early fasciotomy at left lower leg in our case may improve the local reperfusion of affected muscles and prohibit the progression of the ischemic tissue to necrosis.

In the other hand, the type 2 lesions having similar MRI features to those in the recent report [3] showed gradually decreased signal intensity on both T1-weighted and T2-weighted images without contrast enhancement attributed to intra-muscular fibrosis and/or old hemorrhage, i.e. hemosiderin deposition.

We have discussed MRI can predict the possible acute cause of rhabdomyolysis is reversible or irreversible.
The reversible region is expected to recover undergoing adequate treatment to stop further muscle destruction, and the irreversible region meant possible intra-muscular fibrosis and/or old hemorrhage, and a high possibility of losing normal muscular function afterwards. Rehabilitation should be arranged to the correspondent affecting muscle groups and strengthen the complementary muscles.

CONCLUSION

In conclusion, MRI played an important role in the assessment of the severity and extent of the affected muscles in rhabdomyolysis. Two distinctive forms of rhabdomyolysis is demonstrated on MRI, with the type 1 lesion, representing the reversible, and the type 2 present with poor outcome since severe myonecrosis and fibrosis.

REFERENCES